

## Hematuria Following Ingestion of Pyribenzamine Hydrochloride

### Report of a Case

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WITH the widespread use of antihistaminic drugs, reports of toxic side-reactions have been increasing. Gastrointestinal and cerebral reactions predominate. In one of the most recent reports, Wolfson<sup>2</sup> noted urinary system disturbances following ingestion of pyribenzamine hydrochloride, with no gastric or cerebral reactions. In that case, the symptoms of diminishing urinary stream and increased bladder pressure, in the absence of any abnormalities in the urinary elements, indicated transient obstruction, probably in the region of the bladder neck.

A patient observed by the author had gross blood in the urine, associated with bladder pain, frequency and nocturia, after taking pyribenzamine hydrochloride.

### CASE REPORT

A white male, aged 32, had been under observation and treatment for six months for chronic, non-specific prostatitis. Complete studies had indicated that there was no disease in the kidneys, as evidenced by normal intravenous pyelograms. In cystourethrograms taken several months before no abnormalities had been noted. In an effort to clear the prostatic infection, foci elsewhere were searched for. Chronic ethmoid sinusitis was present; and an otolaryngologist who, in consultation, diagnosed the condition as of allergic origin, treated the patient with sinus washings and then advised pyribenzamine hydrochloride in doses of 50 mg. four times a day. Two days after treatment with the antihistaminic agent was started, gross hematuria occurred. Associated with it were bladder pain, frequency of urination every 20 minutes during the day and hourly at night. The urine was grossly rust-colored, the reaction to a chemical test for blood was strongly positive, and innumerable erythrocytes per high-power field were noted in microscopic examination. Upon physical examination, no other toxic side-reactions were noted and no other condition that might cause the bleeding. The urethra and prostate were normal to palpation, as were the vesicles. Tenderness in the bladder was noted on deep palpation. The pyribenzamine was discontinued and urinary sedatives and antispasmodics were given. The following day, the bleeding was considerably diminished, but the urine still contained 50 to 60 erythrocytes per high-power field. There were no leukocytes. Reaction to a test for blood was 3+, and for albumin 2+. Within five days the symptoms had fully abated and the urine was microscopically normal.

### DISCUSSION

The case reported is another in which urinary disturbances were noted following administration of pyribenzamine hydrochloride. In the previously reported case,<sup>1</sup> dysuria and increased frequency ceased in one day following discontinuance of the drug. In the present case, however, there was true hemorrhagic cystitis without actual infection. The mechanism was probably purely an irritative phenomenon. The author believes it likely that the excretion of the drug in the urine, in sensitive subjects, causes edema and exudation of the bladder mucosa, especially in the trigone region, much in the way that the formaldehyde of urotropin breakdown causes these conditions in the bladder of sensitive subjects. Wolfson<sup>3</sup> in his report indicated that "the antihistaminic may possess a spasmogenic property heretofore unrealized."

It would seem to be worth while for physicians giving pyribenzamine hydrochloride to question patients more fre-

quently and more carefully with regard to urinary discomforts so that these complications may be avoided.

### SUMMARY

The case reported is one in which gross blood appeared in the urine, with frequency, nocturia and bladder pain, following ingestion of pyribenzamine hydrochloride.

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### REFERENCES

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3. Wolfson, S. A.: Urinary obstruction due to tripeleminamine hydrochloride, *J.A.M.A.*, 140:958, July 16, 1949.

## Familial Occurrence of Polycythemia and Leukemia

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THE existence of polycythemia in two or more members of a single family has been reported by several observers, and in 1933 the cases previously reported in the literature were reviewed by Spodaro and Forkner,<sup>15</sup> who found six studies of what they believed to be proven familial polycythemia. These examples are cited briefly below.

Bernstein<sup>2</sup> described a polycythemic patient whose son had an erythrocyte count of 7.5 million and whose father, who died at the age of 47, had had a high color. In a family reported upon by Doll and Rothschild,<sup>5</sup> five members had Huntington's chorea; two of the five also had erythrocyte counts of six million or over, and one had hypertension. Another member also had hypertension, and the erythrocyte count was five million. Engelking<sup>6</sup> described polycythemic traits in three generations of a family; in the last generation, six of 13 children had erythrocyte counts ranging from 8 million to 13.6 million with hemoglobin value between 140 per cent and 185 per cent. In two the spleen was palpably enlarged, and in three there was clubbing of the fingers. Infantilism was coexistent with polycythemia in five of the children. Kretschmer<sup>10</sup> recorded polycythemia in three children in a single family. The father was not examined, but the mother was normal. In two of the three children, the clubbing of the fingers and toes and the cardiac findings led to suspicion that the polycythemia was secondary to congenital heart lesions. In the other instances, no such associated constitutional abnormalities were present. Tancre<sup>16</sup> reported a patient with an erythrocyte count of 14.2 million, hemoglobin value of 17.8 per cent and leukocyte count of 18,300. This patient had a sister with erythrocyte count of 6.1 million but no symptoms. Weil and Stiefel<sup>18</sup> described a patient with erythrocyte count ranging from 5.5 million to 7 million and hemoglobin values from 115 per cent to 130 per cent. Cyanosis of the lips and extremities was noted, and the liver and spleen were palpably enlarged. The patient's brother was cyanotic, and the erythrocyte count was 6.44 million. He had no symptoms, and the liver and spleen were not enlarged.

In addition to a review of the previously reported cases of familial polycythemia, Spodaro and Forkner<sup>15</sup> described a family in which splenomegaly as well as high erythrocyte

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